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**Effects of high intensity / low volume and low intensity / high volume isokinetic
resistance exercise on post-exercise glucose tolerance**

by

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LaCrosse, WI 2000

Presented in partial fulfillment of the requirements

for the degree of

Master of Science

University of Montana

Missoula, MT 2002

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Effects of high intensity / low volume and low intensity / high volume isokinetic resistance exercise on post-exercise glucose tolerance

Director: Brent C. Ruby, Ph.D.

The purpose of this study was to determine the effects of high intensity/low volume (HILV) and low intensity/high volume (LIHV) isokinetic resistance exercise on post-exercise glucose tolerance. Subjects ($n = 10$) performed two separate isokinetic resistance exercise trials (HILV and LIHV) of reciprocal concentric knee flexion and knee extension in a fasted state. Each bout was followed by a 45-min oral glucose ($1.8\text{g}\cdot\text{kg FFM}^{-1}$) tolerance test (OGTT). Blood samples were obtained every 15 min to determine glucose and insulin concentrations. There was no difference in total work (J) between the two trials ($p = .229$). Blood glucose was significantly higher at all time points compared to time 0 following the LIHV trial ($p < 0.05$). Following the HILV trial, blood glucose was significantly elevated at 15 and 30 min ($p < 0.05$) but returned to resting values by 45 min. Insulin concentration was significantly elevated following both trials at all time points ($p < 0.05$). Blood glucose and insulin were significantly higher following the LIHV at 30 and 45 min compared to the HILV trial ($p < 0.05$). These results demonstrate that although the total work output was similar across trials, high intensity muscle contraction is associated with an enhanced normalization of glucose homeostasis following a large post-exercise oral glucose feed.

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Table of Contents

Chapter One: Introduction	1
Introduction	1
Problem	3
Research Hypothesis	3
Significance of the Study	3
Rationale of the Study	4
Limitations	5
Delimitations	5
Definition of Terms	6
 Chapter Two: Review of Related Literature	 8
Cardiovascular endurance studies on glycogen resynthesis and glucose tolerance	8
Studies on the mode, timing, and type of carbohydrate administration	11
Resistance training studies on glycogen resynthesis and glucose tolerance	15
GLUT-4 transporter role	17
Effects of exercise training on diabetes mellitus	19
 Chapter Three: Methodology	 23
Setting and Subjects	23
Descriptive Data	23
Exercise Testing	24
Average Peak Force Test	24
Control and Exercise Trials	25
Oral Glucose Tolerance Tests	26
Blood Sampling and Metabolite Assays	27
Research Design and Statistical Procedures	27
 Chapter Four: Results	 29
Chapter Five: Discussion	31
References	35
 Appendix I	 39
Institutional Review Board	40
Informed Consent Form	41
Medical History Questionnaire	45
Physical Activity Related Questionnaire (PAR-Q)	46
 Appendix II	 47
Data Sheets	
 Appendix III	 50
Statistical Results	

Chapter One: Introduction

Introduction

It has been well established the physiological benefits of resistance training.

Improvements in muscular strength (Ballor et al., 1996; Durak et al., 1990; Geliebter et al., Hurley et al., 1988; Marcinik et al., 1991; Miller et al., 1984; Snow-Harter et al., 1992) bone mineral density (Snow-Harter et al., 1992), blood lipid profiles (Honkola et al., 1997; Hurley et al., 1988), and fat free mass (Miller et al., 1984) are the most common benefits attributed to resistance training. Furthermore, resistance training has also been shown to maintain lean body weight under dieting conditions (Bryner et al., 1999), improve endurance performance (Marcinik et al., 1991), and glycemic control (Durak et al., 1990; Eriksson et al., 1997; Honkola et al., 1997; Miller et al., 1984).

Control of blood glucose is the primary focus of this study and is a major treatment goal in type II diabetics.

Several studies have demonstrated increased glucose uptake and subsequent muscle glycogen resynthesis following resistance exercise (Pascoe et al., 1993; Roy et al., 1997, Roy and Tarnopolsky, 1998). The pathology behind type II diabetes involves both peripheral insulin resistance and a decreased insulin secretion thus resulting in hyperglycemia. Resistance exercise would theoretically improve glucose metabolic profiles in type II diabetes patients. Eriksson et al. (1997) and Honkola et al. (1997) demonstrated improvements in long-term glycemic control (glycosylated hemoglobin) following a circuit resistance training program in type II diabetics. In addition, a study by Durak et al. (1990) showed improvements in glycemic control in type I diabetics

following a resistance training program. Other authors found a reduced plasma insulin response to glucose load following a resistance training program in normal subjects (Hurley et al., 1988; Miller et al. 1984). Thus, resistance exercise has benefits that would be applicable towards treatment of type II diabetes. The mechanism responsible for improved glycemic control seems to be increased GLUT-4 transporter translocation.

Review articles conducted by Barnard and Youngren (1992), Borghouts and Keizer (2000), and Hayashi et al. (1997) suggest that contraction can recruit GLUT-4 to the plasma membrane and transverse tubules independent of insulin. Other studies using rats had similar findings (Constable et al., 1988; Goodyear et al., 1990; Ploug et al. 1997). In contrast, a few studies demonstrate decreased GLUT-4 protein content and insulin following eccentric exercise (Asp et al., 1995; Asp et al., 1996; Asp et al., 1997). It has been suggested that two distinct intracellular locations of glucose transporters exist, one that responds to exercise and one that responds to insulin (Brozinick et al., 1994).

Several studies have investigated post-exercise glucose uptake immediately following endurance exercise (Bergstrom and Hultman, 1967; Blom et al., 1987; Blom, 1989; Ivy et al., 1988; Ivy et al., 1988; MacDougall et al., 1977; Maehlum et al., 1978; Reed et al., 1989; Zawadzki et al., 1992). Furthermore, studies by Bonen et al. (1998) and Young et al. (1989) compared the effects of intensity of exercise on glucose uptake. Both of the studies found no difference in glucose tolerance between the two intensities studied. In contrast, few studies have examined glucose uptake following resistance exercise (Pascoe et al., 1993; Roy et al, 1997; Roy and Tarnopolsky, 1998) where as to our knowledge,

none have investigated the effect of exercise intensity on glucose uptake. Further research is warranted in this area.

Problem

The purpose of this study is to determine the effects of high intensity/low volume and low intensity/high volume isokinetic resistance exercise on post-exercise glucose tolerance. The study will examine the change in blood glucose and insulin levels using an oral glucose tolerance test (OGTT) during a control session and immediately post-exercise. The primary variables of interest will be blood glucose and insulin levels post-exercise.

Research Hypothesis

Hypothesis One

There will be an improved blood glucose and insulin response post-exercise following the high intensity / low volume trial.

Significance of the Study

Several studies have examined the effect of a resistance training program on glucose metabolism (Durak et al., 1997; Eriksson et al., 1997, Honkola et al, 1997; Hurley et al., 1988; Miller et al., 1984). Others have investigated the effect of timing and mode of supplementation post-exercise on muscle glycogen resynthesis (Pascoe et al., 1993; Roy et al., 1997; Roy and Tarnopolsky, 1998). This will be the first study to compare glucose tolerance following resistance exercise of different exercise intensities.

Only two studies using cardiovascular endurance exercise have compared glucose tolerance post-exercise. Each study demonstrated similar benefits of high and low intensity exercise on glucose tolerance (Bonen et al., 1998; Young et al., 1989). Results of this study may add to development of guidelines for type II diabetics who wish to increase muscular strength. Furthermore, it may aid individuals in choosing the amount of supplement needed following a particular resistance training session.

Rationale for the Study

The comparison of glucose tolerance following resistance exercise of different intensities is necessary for a more complete understanding of the mechanisms that cause glucose uptake without insulin. Contraction stimulated GLUT-4 translocation appears to be the reason for enhanced glucose uptake following exercise. It may be possible that the more intense the contraction, the higher the number of GLUT-4 translocation. If this occurs, an increased glucose uptake without insulin would result. In addition to the acute effects of resistance exercise on glucose tolerance, Miller et al. (1984) and Szczypaczewska et al. (1989) demonstrated the long-term effects of resistance training where trained subjects had reduced insulin responses. Furthermore, Honkola et al. (1997) showed improvement in long-term glycemic control following a resistance training program in subjects with type II diabetes. In essence, it would be beneficial for type II diabetics to perform resistance exercise at an intensity that results in the most improved glucose tolerance.

Limitations

i/ Non-randomized sample. The subjects in the study were not randomly selected, they were selected on the basis of their previous weight training experience.

ii/ Instrumentation. As with most experimental research, there is an inherent error associated with the use of equipment. This error will be minimized by using regularly calibrated equipment and properly trained researchers.

Delimitations

i/ Type of subjects. The type of subjects used for the study have had at least one year of previous weight training experience. This was established to minimize soreness possibly resulting from the exercise trials and to enhance familiarization of subjects with both the equipment and the exercise protocol. Subjects with Type I or Type II diabetes as well as bleeding disorders were excluded from the study.

ii/ Males only. Volunteer male subjects were used in this study. Males were selected so as to not encounter menstrual cycle variations in addition to other gender specific issues.

iii/ Age of subjects. The age of subjects in the study ranged from 18-30 years of age. This range was chosen for simplicity so as to factor out special populations.

iv/ Specific exercise protocols. The low intensity / high volume and high intensity / low volume exercise protocols were chosen to provide enough stimulus to deplete muscle glycogen stores and to replicate a typical exercise session for muscular strength and endurance in a fitness facility.

v/ Specific exercise mode. Isokinetic resistance exercise will be the mode of the exercise in this study. This was selected in order to quantify total work (J) between exercise trials and to keep the speed of movement and range of movement constant.

vi/ Specific exercise selection. Reciprocal concentric knee flexion and extension were chosen for this study. These exercises were chosen due to the large muscle mass of the quadriceps and hamstring muscles used in the movement. Eccentric exercise was not selected as part of the exercise mode due to its detrimental effects on glucose uptake and muscle glycogen resynthesis (Asp et al., 1995; Asp et al., 1996; Asp et al., 1997).

Definition of Terms

GLUT-4 transporters: one isoform of transporter proteins present within the muscle cell. These translocate from the interior to the outer membrane of the muscle cell resulting in an increase in glucose transport.

Isokinetic exercise: a method that relies on the use of a machine to control the speed of movement over a range of movement. The device enables the control of angular velocity with a reaction force throughout the full joint range of motion.

Oral glucose tolerance test (OGTT): involves orally ingesting some type of glucose solution to examine glucose kinetics.

Type II diabetes (Non-insulin dependent or adult onset): a chronic metabolic disease characterized by peripheral and hepatic insulin resistance as well as impaired insulin secretion.

Chapter Two: Review of Literature

Cardiovascular endurance studies on glucose tolerance and glycogen resynthesis

The importance of exercise and post-exercise supplementation has gained significant popularity in both the general and athletic populations. A large majority of previous research focuses on the use of post-exercise carbohydrate supplements. It is through this carbohydrate supplementation that muscle glycogen resynthesis is enhanced and permits the muscular system to be in an anabolic state.

Maehlum and Hermansen (1978) demonstrated the effect of a 12-hour fast following muscle glycogen depleting exercise. Following an initial fast (12-14 hours), subjects ($n=5$) exercised at 70% VO_{2max} until exhaustion, which was again followed by a 12-hour fast. Muscle biopsies and blood samples were collected at 2, 4, 6, 9 and 12 hours into recovery. No change in immunoreactive insulin or plasma glucose concentrations occurred throughout the recovery period. However, muscle glycogen increased during the first 4 hours of recovery and then remained the same during the next 8 hours.

MacDougall et al. (1977) examined the time course for muscle glycogen repletion following high-intensity intermittent exercise (140% VO_{2max}) on a cycle ergometer. Muscle biopsies were conducted at rest, immediately post-exercise and at 2, 5, 12, and 24 hours after exercise. Subjects consumed identical lunch and supper diets following the 2-hour biopsy where 3 of the subjects consumed an additional carbohydrate supplement (357 g). Glycogen resynthesis was most rapid over the first 5 hours after exercise and

after 24 hours all subjects had regained or exceeded their pre-exercise values for muscle glycogen concentration. No differences were found in the rates of muscle glycogen repletion between the carbohydrate-loaded subjects and those on the normal mixed diet.

Maehlum et al. (1978) investigated splanchnic glucose output and muscle glycogen concentration following two bouts of exhaustive bicycle exercise. Six subjects exercised in the morning prior to glucose ingestion where five subjects performed the identical exercise protocol 14-15 hours before glucose administration. Splanchnic glucose output increased to values 50-300% greater than in controls for the exercised groups. No differences were found in average rates of muscle glycogen synthesis between the exercised groups.

Blom (1989) compared post-exercise muscle glycogen synthesis after oral and intravenous glucose administration. In the first series (OG), 1.4, 0.7, and 0.7 g/kg body mass were given in oral loads at 0, 1, and 2 hours of a 3-hour post-exercise observation period. In the second series (GC), the glucose clamp technique was used to obtain the same plasma glucose concentration at all time points as in the OG series. The results showed no difference in plasma glucose concentrations and muscle glycogen synthesis between the two groups. In contrast, plasma insulin concentration and arterial-femoral venous plasma glucose concentration difference were significantly different between the two experiments ($p < 0.001$).

A study conducted by Bonen et al. (1998) utilized methodology similar to ours. The author's investigated the effects of low and high intensity (60% and 83% of age-adjusted maximal heart rates) endurance exercise on glucose tolerance immediately and 24 hours post-exercise. Oral glucose tolerance tests (75 g) were conducted during a fasted condition and immediately and 24 hours after each exercise condition. Incremental glucose areas under the curve immediately after exercise were reduced by 16% and 14% for the low and high intensity exercise conditions respectively. A further reduction of 30 and 35% in incremental glucose areas under the curve occurred following the low- and high-intensity exercise conditions at 24 hours post-exercise. No differences in OGTT responses were found between the two exercise intensities at either time point.

Young et al. (1989) evaluated the residual effects of high and low intensity exercise on glucose tolerance and insulin response. Two groups of seven ($n=14$) subjects were tested under three conditions, control, exercise at 40% VO_{2max} , and exercise at 80% VO_{2max} . In the morning, 14 hours post-exercise, an OGTT was conducted following each test. Areas under the glucose concentration curve did not differ between trained and untrained subjects and was not affected by the exercise intensity. Insulin areas under the curve were similar for all conditions in the trained subjects where in the untrained, a 30 and 45% reduction in insulin areas under the curve occurred the day after exercise at 40 and 80% VO_{2max} respectively.

These studies demonstrate the beneficial effects of cardiovascular endurance exercise on blood glucose and insulin. In addition, they provide evidence towards the need for some form of carbohydrate supplementation post-exercise.

Studies on the mode, timing, and type of carbohydrate administration

Numerous studies have attempted to determine the optimal method for increasing muscle glycogen stores following exercise. The majority of the scientific literature has utilized an overnight or 12-hour fast plus endurance exercise to deplete muscle glycogen stores in order to answer this question. Post-exercise carbohydrate supplementation seems to be the most effective method for enhancing muscle glycogen resynthesis

Blom et al. (1987) compared the effects of different amounts of glucose loads as well as sucrose and fructose loads on glycogen resynthesis following bicycle ergometer exercise. Subjects (n=27) were divided into five groups where they ingested 0.35, 0.7, or 1.4 g/kg body weight of glucose or 0.7g/kg of sucrose or fructose orally at 0, 2, and 4 hours post-exercise. Results of the study demonstrate higher muscle glycogen resynthesis when glucose dosages of 0.7 or 1.4g/kg body weight are consumed compared to 0.35g/kg. However, no difference was found in muscle glycogen synthesis rates between the two larger doses of 0.7 and 1.4g/kg. Furthermore, both glucose and sucrose ingestion, at .7 g/kg, had a significantly stronger effect on muscle glycogen resynthesis than fructose.

Ivy et al. (1988) performed a similar study to Blom's where he examined post-exercise muscle glycogen storage rates after ingestion of a glucose polymer solution amounting to

1.5 or 3.0g/kg body weight. Either carbohydrate supplement was ingested immediately and 2 hours post-exercise. No additional benefit was found in regards to glycogen storage rates when the amount of carbohydrate supplement was doubled from 1.5 to 3.0g/kg body weight. However, ingestion of 1.5g glucose/kg does elevate glycogen storage rates if the supplement is provided immediately and at 2-hour intervals following exercise.

Bergstrom and Hultman (1967) conducted one of the earlier studies examining post-exercise resynthesis of muscle glycogen through administration of glucose or fructose infusion. Two exercised and two non-exercised groups served as subjects where 4g/kg body weight of glucose or fructose were infused post-exercise or after a resting (control) condition. No differences in muscle glycogen were found in subjects who had not exercised before infusion of both glucose and fructose. However, resynthesis of muscle glycogen was significantly higher after infusion of glucose in exercised subjects than after fructose infusion.

A study conducted by Reed et al. (1989) compared muscle glycogen storage post-exercise using liquid or solid carbohydrate feedings, or an intravenous glucose infusion. Subjects (n=8) received one of the three treatments in random order following a 2-hour exercise session for a 4-hour recovery period. Differences in post-exercise plasma insulin and glucose responses were found between the three treatments however no differences in muscle glycogen storage rates were found during the first or second 2 hours of recovery.

Ivy et al. (1988) conducted another study on the effect of timing of ingestion of a carbohydrate supplement post-exercise on the rate of muscle glycogen synthesis. Subjects (n=12) ingested 2g/kg body weight of a glucose polymer solution immediately after or 2 hours after 70 minutes of cycle ergometry exercise. Results of the study demonstrate a 45% slower rate of muscle glycogen storage in the group who ingested the carbohydrate supplement 2 hours post-exercise.

Zawadzki et al. (1992) examined differences in muscle glycogen storage rates between carbohydrate, carbohydrate-protein, and protein supplementation following endurance exercise. Nine trained male cyclists received a supplement containing 112.0g carbohydrate, 40.7g protein, or 112.0g carbohydrate and 40.7g protein on three random experiments immediately and 2 hours after riding on a cycle ergometer. Plasma glucose and insulin responses as well as muscle glycogen storage rates were collected during the 4-hour recovery period. In the recovery period, the plasma glucose response following the carbohydrate treatment was significantly higher than the other two treatments where the plasma insulin response for the carbohydrate-protein treatment was higher. Furthermore, muscle glycogen storage rates for the carbohydrate-protein treatment were 38% faster than the carbohydrate treatment.

In contrast to the findings of Zawadzki et al. (1992), Roy and Tarnopolsky (1998) found similar rates of muscle glycogen resynthesis post-exercise after consumption of an isoenergetic carbohydrate (CHO) or carbohydrate/protein/fat (CHO/Pro/fat) formula.

Subjects (n=10) ingested either formula or a placebo following resistance exercise.

Results demonstrated no differences in glucose and insulin areas under the curve or rates of muscle glycogen resynthesis between the CHO or CHO/Pro/fat trials. However, both trials were significantly greater for glucose and insulin areas under the curve and muscle glycogen resynthesis than the placebo trial.

Roy et al. (1997) investigated the effects of carbohydrate supplementation immediately after resistance exercise on protein metabolism. Subjects (n=8) consumed a placebo or carbohydrate supplement (1g/kg body weight) immediately and 1-hour post-exercise. Total areas under the insulin and glucose curves were significantly higher for the carbohydrate compared to the placebo condition. Furthermore, carbohydrate supplementation post-exercise was found to promote a more positive body protein balance.

Altogether, a review article by Friedman et al. (1991), discusses factors including the mode, timing, and amount of carbohydrate needed post-exercise. Following moderate intensity exercise, complete muscle glycogen synthesis generally occurs within 24 hours if approximately 500 to 700g of carbohydrate are provided. Resynthesis of muscle glycogen is fastest during the first 2 hours post-exercise where ingestion of 0.7g glucose/kg body weight every 2 hours appears to maximize the resynthesis rate during the first 4 to 6 hours. Ingestion of greater amounts of glucose does not provide any additional benefit towards muscle glycogen resynthesis. In regards to the mode of carbohydrate, glucose or sucrose ingestion enhances muscle glycogen resynthesis rates

more than fructose. Furthermore, ingestion of simple carbohydrate compared to complex increases muscle glycogen content more during the first 4 to 6 hours post-exercise.

Resistance training studies on glucose tolerance and glycogen resynthesis

It has been demonstrated that the effects of resistance training on one's metabolic profile are similar to those produced by cardiovascular endurance exercise. Improvements in glucose tolerance and insulin action as a result of resistance training appear to be independent of changes in $\text{VO}_{2\text{max}}$, body composition, and body fat distribution.

Miller et al. (1984) investigated glucose tolerance and glucose-stimulated insulin responses before and after 10 weeks of isotonic resistance training. Glucose and insulin response areas under the curve were calculated following a standard 100g oral glucose challenge. The strength training program resulted in a significant increase in lean body mass (3.5%), however, no differences were found in the area under the glucose curve pre- to post-training. In contrast, a significant reduction (18.9%) in the area under the insulin response curve did occur post-training.

Pascoe et al. (1993) had untrained subjects ($n=8$) perform two high-intensity resistance exercise trials to examine muscle glycogen resynthesis rates. Subjects ingested either water or carbohydrate (1.4 g/kg body weight) following each trial. Muscle glycogen content was restored to 91% of pre-exercise levels after 6 hours of recovery when carbohydrate was administered. In contrast, muscle glycogen content without caloric consumption was restored to 75% although the level of depletion was 71%.

A study by Szczypaczewska et al. (1989) compared blood glucose and insulin responses from a fasted 100g OGTT in body builders, lean untrained subjects (controls), and mildly obese men with a lean body mass similar to the body builders. Blood sampling for glucose and insulin concentrations were taken 30, 60, 90, and 120 minutes after the ingestion of glucose. The sum of glucose concentrations (30 to 120 min) were significantly lower in body builders compared to controls. In contrast, the sum of plasma insulin concentrations in obese subjects was significantly greater than that in the controls.

Miller et al. (1994) tested previously sedentary 50- to 65- year old men (n=11) using an OGTT following 16 weeks of strength training. Post-training, the group as a whole had no differences in fasting plasma glucose levels or glucose levels during an OGTT. In contrast, both fasting plasma insulin levels and insulin levels during the OGTT were significantly lower post-training.

Hernandez et al. (2000) measured the rates of protein synthesis and glucose uptake after acute resistance exercise in male Sprague-Dawley rats. Rates of protein synthesis and glucose uptake were studied in vivo 1, 3, 6, 12 and 24 hours after resistance exercise (n=7) or in a control group (n=7). Glucose uptake initially decreased at 3 hours after exercise and then increased at 6 hours where protein synthesis did not change for at least 6 hours after exercise. Furthermore, both protein synthesis and glucose uptake were elevated at 12 hours but only protein synthesis was elevated at 24 hours post-exercise.

In 2002, Hurlbut et al. conducted a comparison study on young men (n=12) and women (n=9) and older men (n=12) and women (n=9) following 6 months of strength training. An OGTT was conducted pre-training and 24 hours after the last training session. No differences were found in fasting glucose or areas under the glucose curve pre- to post-training for men and women. However, higher glucose areas were noted for older women after training. In contrast, the strength training program resulted in reduced fasting insulin concentrations in both young and older men. Also, young men had a significant (21%) reduction in total insulin area under the curve where there was a trend towards significance (11%) in older men. No improvement in insulin responses were found for young or older women.

The primary effect of resistance exercise on glucose homeostasis appears to lead towards improved insulin sensitivity as opposed to glucose tolerance. It is evident that muscle contraction / exercise, as demonstrated by previous research in cardiovascular endurance and resistance exercise, is the mechanism by which one's metabolic profile is improved.

GLUT-4 transporter role

It has been demonstrated that the rate-limiting step in glucose metabolism is mediated by GLUT-4 transporter proteins. The increased glucose uptake involves the translocation of the GLUT-4 transporter from an intracellular location to the cell surface. Previous research has shown that two distinct pools of GLUT-4 transporters exist in skeletal muscle, one sensitive to insulin, and one sensitive to muscle contraction / exercise (Brozinick et al. 1994 and Etgen et al. 1997). Authors Brozinick et al. (1994) and Etgen

et al. (1997) demonstrated that insulin plus contraction increased glucose uptake above that of insulin or contraction alone. In contrast, Constable et al. (1988) and Goodyear et al. (1990) found that the effects of insulin and contraction to increase muscle glucose transport are not additive. However, Constable et al. (1988) may have used a nonphysiologically high concentration of insulin (20,000 μ U/ml) to study in vivo actions of insulin.

Previous research on rats examined the effects of eccentric contractions on GLUT-4 protein content and insulin action (Asp et al. 1995, Asp et al. 1996, Asp et al. 1997). One of the studies demonstrated a decreased GLUT-4 protein and glycogen concentration content following eccentric exercise in white and red gastrocnemius muscles compared to controls (Asp et al. 1995). In another study using eccentric contractions, the authors demonstrated an impaired insulin-stimulated glucose transport at maximal insulin concentrations (Asp et al. 1996). Finally, the authors added to their previous findings using the conscious rat and demonstrated impaired insulin action on local muscle glucose uptake and glycogen synthesis. Thus, it is evident that eccentric contractions impair the ability of GLUT-4 transporter proteins to increase glucose uptake and subsequent muscle glycogen resynthesis.

Although it has been demonstrated the effects of exercise / muscle contraction on GLUT-4 transporter proteins, the mechanism by which glucose uptake is enhanced remains unclear. Review articles by Hayashi et al. (1997) and Ryder et al. (2001) identify possible mechanisms by which muscle contraction may activate glucose transport. These

mechanisms appear to involve the initiation of a contraction signal. Possible reasons for this initiation could include: release of calcium, activation of AMP-activated protein kinase, and nitric oxide activity.

Effects of exercise training on diabetes mellitus

Both cardiovascular and resistance exercise have shown to improve the metabolic profile of both diabetic and non-diabetic subjects. It is evident that the most likely mechanism for this improvement is a result of contraction induced GLUT-4 transporter translocation. Specifically, the pathology behind type II diabetes involves both peripheral insulin resistance and a decreased insulin secretion thus resulting in hyperglycemia. Thus the use of resistance exercise would be a major treatment goal for type II diabetics.

Maehlum (1978) examined post-exercise muscle glycogen repletion in non-diabetic and diabetic subjects. Subjects bicycled at 60 to 70% of VO_{2max} until exhaustion which was immediately followed by infusion of 0.5g glucose/kg body weight. Muscle biopsies and blood samples were taken before and after exercise at various time points. No differences in muscle glycogen content or glycogen synthesis rate occurred between the two groups. However, both the diabetic and non-diabetic subjects significantly increased muscle glycogen content 65 minutes after the glucose infusion in the post-exercise recovery period.

Allenberg et al (1988) investigated the effects of endurance exercise training on glucohomeostasis in subjects with type II diabetes mellitus. Seven middle-aged men

performed bicycle exercise 3 days per week for a minimum of 10 weeks. Prior to the training period and 72 hours after the last training session, subjects underwent an OGTT (75g glucose solution). Results of the study demonstrate that training did not change glucose and insulin areas under the curve, fasting blood glucose, or levels of glycosylated hemoglobin.

A study by Braun et al. (1995) compared the effects of several bouts of low (50% $\text{VO}_{2\text{max}}$) and high intensity (75% $\text{VO}_{2\text{max}}$) exercise on insulin sensitivity. The duration of exercise was adjusted to make energy expenditure equal in both exercise conditions. Eight obese women with NIDDM served as subjects. Insulin sensitivity during a glucose/insulin infusion was increased after exercise compared to a non-exercise condition however no differences in post-exercise insulin sensitivity occurred between low and high intensity exercise.

Eriksson et al. (1997) investigated the effects of circuit resistance training on long-term glycemic control in subjects with non-insulin diabetes mellitus. Glycosylated hemoglobin $\text{A}_{1\text{C}}$ ($\text{HbA}_{1\text{C}}$) along with other clinical and metabolic parameters were assessed prior to and 5 to 7 days after 3 months of circuit resistance training. Results of the study demonstrated improvement in long-term glycemic control where $\text{HbA}_{1\text{C}}$ fell from 8.8 to 8.2% ($p < 0.05$). Furthermore, there was a strong negative correlation ($r = -0.73$; $p < 0.05$) between total knee extensor cross-sectional area and $\text{HbA}_{1\text{C}}$ compared to baseline ($r = -0.58$; $p = 0.13$).

A similar study by Honkola et al. (1997) also examined long-term glycaemic (HbA_{1c}) in type II diabetics. Differences between this study and Eriksson's (1997) were the inclusion of a control group and the circuit resistance training program was extended to 5 months. In contrast to Eriksson's findings, result showed an improvement in total cholesterol (12%), LDL-cholesterol (14%), and triglycerides (20%). Glycosylated haemoglobin A_{1c} was not different from pre- to post-training in the intervention group where there was a significant difference in the change of HbA_{1c} (0.5%) between groups.

Fluckey et al. (1994) investigated glucose and insulin responses 18 hours after a single bout of resistance exercise in young control subjects (n=7), older patients with NIDDM (n=7), and older age-matched control subjects (n=3). A 75g OGTT was administered first as a control trial (pre-exercise) and then 18 hours post-exercise. For glucose area under the curve, no differences were found from pre- to post-exercise in all groups. However, the insulin area under the curve response post-exercise was significantly lower in the young control and NIDDM groups where there was no change in the age-matched controls.

A study by Smutok et al. (1994) compared the effects of 20 weeks of strength training, aerobic training, or no exercise on men with either non-insulin-dependent diabetes mellitus (n=8), impaired glucose tolerance (n=12), or hyperinsulinemia with normal glucose tolerance (n=6). Two OGTT's were conducted pre- and post-training and demonstrated decreased plasma glucose and insulin areas under the OGTT curve following both strength and aerobic training. No changes were observed in the control

group. In addition, there were no significant differences in OGTT results between the strength and aerobic training groups.

Durak et al. (1990) studied the effects of 10 weeks of resistance training on glycemic control in type I diabetic men (n=8). Following the training program, glycosylated hemoglobin dropped 1.1% from pre-training levels. In addition, self-monitored blood glucose values taken before and after each training session showed a decrease from 7.85 ± 3.13 to 7.05 ± 2.91 mM ($p = 0.0001$).

As demonstrated by previous research, both resistance and aerobic exercise provide beneficial effects for type II diabetics. These effects include: improved glucose tolerance and insulin sensitivity and possibly decreased fasting plasma glucose and insulin levels. In essence, the prescription of resistance and/or aerobic exercise for a person with type II diabetes would be a significant treatment modality.

Chapter Three: Methodology

Setting

All physiological testing was conducted in the Outpatient Physical Therapy Clinic, St. Patrick Hospital and Health Sciences Center, the Human Performance Laboratory, Department of Health and Human Performance, McGill Hall #121, and the Pharmaceutical Sciences Research Lab # 61, Skaggs Building.

Subjects

A total of 10 college-aged males served as subjects for the study. Each subject had at least one year of recreational resistance training experience and had currently been involved in a resistance-training program for a minimum of one month. Questionnaires regarding subjects' previous resistance training experience, medical history, and physical activity levels were assessed before testing could occur. Subjects with Type I or Type II diabetes or bleeding disorders were excluded from the study. In addition, each subject completed a University of Montana IRB-approved informed consent form prior to participation.

Descriptive Data

Data was collected to determine each subject's height, weight, age, body composition, and previous resistance training experience. Height (cm) was determined using a conventional stadiometer where weight was measured with a calibrated digital scale (Toledo Model 8139, Worthington, OH). Body composition, fat mass (FM), and fat free

mass (FFM) was assessed by hydrostatic weighing. Residual lung volume was calculated using a Helium dilution technique. Percent body fat was calculated from body density using the Siri equation (1961).

Exercise Testing

All muscle testing was performed using the Kinetic Communicator (KIN-COM, Chattecx Corp. Chattanooga, TN), a computer controlled isokinetic dynamometer.

Average Peak Force Test

Each subject participated in a session to determine each subjects Average Peak Force (N) during isokinetic movements at speeds of 60° and 180°/sec through a range of 80° (knee extension through flexion). Prior to testing, subjects were allowed to warm up on a cycle ergometer at a self selected pace for as long as they desired but for a minimum of 5 minutes. Goniometry was used to acquire the desired range of motion. Subjects performed the Average Peak Force test first at 60°/sec then at 180°/sec. On both extremities, subjects performed 2 sets of 5 and 1 set of 3 repetitions: one set at approximately 50% of maximal effort, one set at approximately 75% of maximal effort, and the final set at 100% of maximal effort. Subjects were allowed full recovery between sets. For the speeds of 60°/sec and 180°/sec, the Average Peak Force (N) of the final set was used as the Average Peak Force. For data analysis the absolute Average Peak Force for each subject was converted into a relative value by dividing the Average Peak Force by their FFM.

Control Trial (CT)

Procedures for this trial involved subjects resting in the supine position for the duration of the OGTT. Subjects were allowed to read, listen to music, or watch a video during this time.

High intensity/low volume exercise trial (HILV)

This trial consisted of subjects performing 10 sets of 5 repetitions of concentric knee extension / knee flexion exercise. Prior to the trial, subjects were allowed to warm up on a cycle ergometer at a self selected pace for 5 minutes. One repetition was considered a full concentric extension and concentric flexion of the knee joint within a range of 80°.

Speed of the motion was set at 60°/sec with 2-minute rest intervals between sets.

Subjects completed 5 sets on each extremity in an alternating manner until 10 sets was reached. Approximately 4-5 minutes was permitted when alternating extremities.

Selection of the extremity to perform the exercise first was done randomly. Total work in joules (J) was provided by the KIN-COM after completion of each set and the sum was calculated manually to quantify total work (J) for the entire session. In addition, the Mean Peak Force from each set was averaged to determine the Mean Peak Force for the entire session for knee extension and flexion on both extremities. For data analysis the absolute Mean Peak Force for each subject was converted into a relative value by dividing the Mean Peak Force by their FFM.

Low intensity/high volume exercise trial (LIHV)

This trial has identical components as the HILV trial with the following exceptions.

Subjects would perform 4 sets of 15 concentric knee extension / knee flexion exercise.

Speed of the motion was set at 180°/sec with 2-minute rest intervals between sets and 4 to 5 minutes when alternating extremities. Other requirements for this trial are the same as the HILV trial.

Order of the trials

The control trial was conducted prior to the HILV and LIHV trials which were done at least 4 days apart in random order. Subjects were not allowed to engage in any strenuous exercise 24 hours prior to any of the three trials. The Average Peak Force trial was conducted at any time prior to, in between, or after as long as it was not within 24 hours of any of the three trials. Strenuous exercise includes: resistance exercise, running, stairclimbing, biking, high intensity walking, or any other form of exercise causing a modest elevation in heart rate. The total work from either the HILV or LIHV trial, whichever was performed first, was used as a stopping point for the following trial to be completed. This was done to normalize the total work between trials.

Oral Glucose Tolerance Test's (OGTT)

Each subject received three OGTT throughout the duration of the study. The first test was performed in a resting condition following a 12-hour overnight fast which served as a control trial (CT). Blood samples were taken at time = 0 and every 15 minutes thereafter following consumption of a standard glucose drink (1.8g/kg/FFM). The second and third OGTT were conducted following the HILV and LIHV exercise trials. Subjects

reported to the lab following a 12-hour overnight fast. An indwelling venous catheter was then inserted into an antecubital vein for the pre-trial blood sample. Each subject was then transported to the Outpatient Physical Therapy Clinic to complete one of the two exercise trials in random order. Each exercise trial was immediately followed by the same OGTT procedures as the control trial.

Blood sampling

All blood samples were obtained through antecubital venipuncture. Samples (~5 mL) were collected in test tubes and allowed to clot for at least 15 minutes at room temperature. Samples were spun at approximately 4000 rpm at 4°C for 15 minutes where after the plasma was separated from the whole blood. Samples were removed and stored at -30°C until subsequent analysis.

Metabolite assays

Blood glucose concentration was determined in duplicate using a spectrophotometric technique using a commercially available kit (Procedure UV-47, Sigma Diagnostics, St. Louis, MO). Blood insulin was determined in duplicate using a solid phase two-site enzyme immunoassay (DRG Insulin ELISA, DRG International, Inc., USA).

Research design and statistical procedures

All descriptive data for subjects was presented in terms of mean $\pm$ SD. A Bland Altman Plot and paired samples t-test was used to compare total work (J) between the HILV and LIHV trials. A paired samples t-test was also used to compare Mean Peak Force (N/FFM

(kg)) and Average Peak Force (N/FFM (kg) between the two trials. A repeated measures 2 x 4 design ANOVA was used to examine differences in times 0, 15, 30, and 45 minutes post-exercise. All data analyses were conducted using Microsoft Excel and the SuperAnova statistical package (Abacus Inc, Berkeley, CA). The level of significance was set at an alpha level of 0.05.

Chapter Four: Results

Results

Descriptive statistics including age (years), height (cm), weight (kg), percent body fat, fat free mass (kg), fat mass (kg) and g of carbohydrate (CHO) consumed for the subjects used in the study are provided in Table 1. Average Peak Force (N/FFM (kg)) and Mean Peak Force (N/FFM (kg)) are provided in Table 2. For both Average Peak Force and Mean Peak Force no significant difference was found between the left and right sides, therefore the data was collapsed and is reported by muscle group only. For the Average Peak Force test, significant differences were found between the two intensities (60 deg/sec and 180 deg/sec) for the knee extensors and knee flexors ($p < 0.05$). Significant differences were found for Mean Peak Force between the LIHV and HILV trials for knee extension, but not for flexion of either extremity ($p < 0.05$).

The total work (J) completed for the LIHV and HILV trials is provided in Table 3. Total work for the LIHV and HILV trials was 25784.3 ± 4320.6 J and 25882.1 ± 4439.4 J respectively. The difference in total work between the two trials was not significant ($p = 0.229$). Figure 3 is a Bland Altman Plot showing the difference in total work versus the average total work for both trials. 90% of subjects had less than 1% difference in total work between the LIHV and HILV trials. The remaining 10% had less than 3% difference between trials.

Figures 1 and 2 demonstrate the blood glucose and insulin responses respectively for the time points 0, 15, 30, and 45 min post-exercise. A significant time x trial interaction was

found for both blood glucose ($p = .03$) and insulin ($p = .01$). Blood glucose was significantly higher from time = 0 at all time points during the LIHV trial ($p < 0.05$). During the HILV trial, blood glucose was significantly elevated from time = 0 at 15 and 30 min ($p < 0.05$). Insulin concentration was significantly elevated from time = 0 min at all time points for both trials ($p < 0.05$). Blood glucose and insulin were significantly higher during the LIHV at time = 30 and 45 min compared to the HILV trial ($p < 0.05$).

Chapter Five: Discussion

Discussion

The purpose of this investigation was to determine the effects of high and low intensity resistance exercise on post-exercise glucose tolerance when the total muscle work was kept constant. Results of the study demonstrate an enhanced normalization of blood glucose and insulin following the HILV trial. This is of significance considering the similarity in total work between trials (see Figure 3). The HILV trial was designed to mirror the common prescription for the development of muscular strength including less than 6 repetitions per set performed at a slower speed.

The only previous research examining the effects of exercise intensity on glucose tolerance and insulin responses has used an endurance exercise protocol (11, 12, 44). One study found no differences between intensities in glucose and insulin areas under the curve both immediately after exercise and 24-hours post-exercise (11). A possible limitation to this study is that the authors had subjects exercise at intensities of 60 and 83% of age-adjusted HRmax for the low and high intensity exercise trials. The variance associated with prescribing cardiovascular exercise from heart rates is considerable (1 S.D. = 10-12 b/min) (1). In addition, the high intensity session was performed one week prior to the low intensity session thus possibly causing a residual effect of the previous exercise session.

A study by Young et al. (44) used trained and non-trained subjects and found no differences in plasma glucose and insulin responses to an oral glucose load 14 hours post-

exercise at intensities of 40 and 80% of $\text{VO}_{2\text{max}}$. However, after a single bout of exercise, the non-trained subjects had comparable insulin responses to the trained subjects. Braun et al. (12) had women with non-insulin-dependent diabetes mellitus (NIDDM) exercise at 50 and 75% of $\text{VO}_{2\text{max}}$ and adjusted the duration of exercise so that energy expenditure was equal in both exercise conditions. No differences were found when comparing plasma glucose and insulin responses to a test meal (50% carbohydrate, 20% protein, and 30% fat) 90 min post-exercise.

It has been demonstrated that exercise/muscle contraction mediated glucose transport is proportionate to the magnitude of GLUT-4 translocation to the plasma membrane. Some studies have shown that insulin plus contraction increases glucose uptake above that of insulin or contraction alone (13, 22, 34) whereas others have not (15, 21). However, Constable et al. (15) used a nonphysiological high insulin concentration (20,000 $\mu\text{U/ml}$ for 60 min) to elucidate the actions of insulin. The mechanism by which muscle contraction may activate glucose transport appears to involve the initiation of a contraction signal. Possible reasons for this initiation could include: release of calcium, activation of AMP-activated protein kinase, and nitric oxide activity (23, 39). An explanation for the improved glucose tolerance and insulin response following the HILV trial could be the magnitude of muscle contraction involved. During the HILV exercise trial the subjects were working their knee extensors at an intensity that was significantly greater than during the LIHV exercise trial. Interestingly, there was no significant difference in the work intensity of the knee flexors between the HILV and LIHV exercise trials (see Table 2). Thus, the magnitude of contraction of the knee extensors that

occurred may result in a greater GLUT-4 translocation and subsequent glucose transport. These data indicate that the intensity of contraction is more influential on glucose normalization in comparison to the total muscle work. In addition, a possible link may exist between fast glycolytic fibers and subsequent GLUT-4 translocation. Thus, the improvement in glucose homeostasis shown may be a result of higher intensity contractile force and/or increased fast glycolytic fiber recruitment.

A possible limitation to the study may have been the duration of exercise and rest for each trial. Total exercise and rest time for the HILV trial was 3.8 ± 0.8 and 46.0 ± 7.9 min respectively where total exercise and rest time for the LIHV trial was 2.1 ± 0.4 and 22.8 ± 4.4 min respectively. The difference between the two trials was due to a higher number of sets and rest periods associated with the HILV trial.

The study may have implications towards individuals with insulin resistance or deficiency such as is the case with Type II diabetes. Resistance exercise has already been shown to improve the metabolic profile through either improved glucose tolerance and/or insulin response in people with Type I (16) or Type II diabetes (17, 19, 24, 41). These results demonstrate both improved glucose tolerance and insulin response following a resistance exercise bout similar to the standard for strength development. Therefore, the benefits of higher intensity and lower volume exercise should receive further attention. The effects of fast glycolytic fiber recruitment strategies should also be investigated in terms of the potential to maximize GLUT-4 translocation. In addition, the link between total fiber recruitment and/or fiber type recruitment and GLUT-4 translocation should be

addressed. Interestingly, these results conflict with the guidelines set by the American College of Sports Medicine, which recommends performing resistance exercise in the range of 8 to 12 repetitions (1). Furthermore, these data suggest that decisions regarding the amount of post-exercise carbohydrate supplement are dependent on the nature of the resistance exercise bout (high vs. low intensity) during strength training.

In conclusion, to our knowledge this is the first study to examine blood glucose and insulin levels following two different resistance exercise trials when the total work between the two trials was kept constant. The results demonstrate a more rapid blood glucose normalization in addition to a blunted insulin response following a bout of high intensity / low volume isokinetic resistance exercise. This is most likely a result of enhanced stimulation of contraction sensitive GLUT-4 transporters to the plasma membrane and may indicate fiber dependent and/or ramp GLUT-4 recruitment process during resistance exercise. Further research is needed examining metabolic responses following resistance exercise when variables such as the duration of exercise, mode of resistance exercise, active muscle mass, and type of subjects used are manipulated.

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Appendix I:

Institutional Review Board, Informed Consent, Medical History Questionnaire, PAR-Q

For Internal
Use Only

The University of Montana #7-02
INSTITUTIONAL REVIEW BOARD (IRB)
CHECKLIST

RECEIVED
(Rev. 7/00)
JAN 03 2002

UNIVERSITY OF MONTANA
VICE PRESIDENT FOR RESEARCH

Submit one completed copy of this Checklist, including any required attachments, for each course involving human subjects. The IRB meets monthly to evaluate proposals, and approval is granted for one academic year. See IRB Guidelines and Procedures for details.

Project Director: Brent C. Luby, Ph.D. Dept.: HHP Phone: x 2117

Signature: [Signature] Date: 12/20/01

Co-Director(s): Andrew Miller Dept.: HHP Phone: x 5528
549-8126

Project Title: Effects of high intensity/low volume and low intensity/high volume
isokinetic resistance exercise on post-exercise glucose tolerance.

Project Description: Glucose tolerance will be measured in recreationally
(in nontechnical language) trained subjects during a control trial and following
two bouts of isokinetic resistance exercise.

All investigators on this project must complete the NIH self-study course on protection of human research subjects.

Certification: I/We have completed the course - (Use additional page if necessary)

Signature	Date	Signature	Date
<u>[Signature]</u>	<u>05/01</u>	<u>[Signature]</u>	<u>05/01</u>
<u>[Signature]</u>	<u>05/01</u>		

Students Only:

Faculty Supervisor: [Signature] Dept.: HHP Phone: x2117

Signature: [Signature]
(My signature confirms that I have read the IRB Checklist and attachments and agree that it accurately represents the planned research and that I will supervise this research project.)

For IRB Use Only

IRB Determination:

 Approved Exemption from Review

 Approved by Administrative Review

 X Full IRB Determination:

- Approved
- X Conditional Approval (see attached memo)
- Resubmit Proposal (see attached memo)
- Disapproved (see attached memo)

Signature IRB Chair: [Signature] Date: 1/16/02
(over)

Subject Information and Consent Form

**Human Performance Laboratory
Dept. of Health and Human Performance
The University of Montana**

Title: Effects of high intensity / low volume and low intensity / high volume isokinetic resistance exercise on post-exercise glucose tolerance

Investigators: Brent C. Ruby, Ph.D., McGill Hall, 243-2117
Andrew D. Miller, McGill Hall, 243-5528
James J. Laskin, Ph.D., Skaggs Building, 243-4757

Special Instructions to the Potential Subject:

- This consent form may contain words that are unfamiliar to you. Please feel free to ask any questions about the following material.

Purpose:

- You are being asked to participate in a research study examining glucose uptake following two different trials of resistance training.
- You have been asked to volunteer because you have a minimum of one year of recreational resistance training experience and because you have been engaged in a resistance training program for at least one month.
- The purpose of this study is to determine the effects of high intensity / low volume and low intensity / high volume resistance exercise on post-exercise glucose tolerance.

Procedures:

- If you agree to participate in this research study you will be involved in four testing situations (resting trial, a measure of body composition and one-repetition maximum test, high intensity / low volume trial, low intensity / high volume trial). One of these sessions will take approximately 1 hour while the other three will take approximately 3 hours. The muscle testing will be done using an isokinetic device which is a machine that allows you to control the speed of movement over a range of movement. Twelve subjects will be included in the study.
 - Session 1: body composition and one-repetition maximum tests. The one-repetition maximum test will consist of performing 1 set of 6 to 8 repetitions and 2 sets of 3 to 4 repetitions of knee extension and flexion exercise at slow (1 rep/sec) and fast (2 reps/sec) speeds. Prior to the one-repetition maximum test, body weight in a bathing suit, height, and body composition will be measured. Body composition will be measured using underwater weighing and the helium dilution technique. Underwater (hydrostatic) weighing consists of total immersion in a warm tank (similar to a hot tub) and holding ones' breath for a bout 4 seconds after exhaling as much air as possible. Body

fat will be calculated from the results of hydrostatic weighing. The helium dilution technique requires breathing 4 deep breaths in and out of a bag containing oxygen and helium. There is no associated pain or discomfort with this technique. It will take place in the afternoon and will last approximately one hour.

- Session 2: resting trial. This trial will be conducted after a 12-hour fast and consists of subjects lying in the supine position following the ingestion of a standard glucose drink of about 0.75 L to 1.2 L of 12% solution (25 to 41 oz.). Blood sampling will then occur for approximately 2 to 3 hours until blood glucose returns to baseline.
- Session 3: high intensity / low volume trial. This trial will also be conducted after a 12-hour fast and will consist of performing 10 sets of 5 repetitions of knee extension and flexion exercise at a slow speed (1 rep/sec). It will take approximately 40 minutes to complete the prescribed exercise. Immediately after this trial, the same procedures will be followed as the resting trial. Total test time will be approximately 2 to 3 hours.
- Session 4: low intensity / high volume trial. This trial may be conducted before the high intensity / low volume trial where the order of these two trials will be chosen randomly. It will take approximately 40 minutes to complete the prescribed exercise. This will consist of performing 5 sets of 15 repetitions of knee extension and flexion exercise at a fast speed (2 reps/sec). Immediately after this trial, the same procedures will be followed as the resting trial. Total test time will be approximately 2 to 3 hours.
- Blood sampling will take place before and after the resting and exercise trials but will not take place during the one-repetition maximum test. An indwelling venous catheter (18 to 20 gauge needle 1.16" long) will be inserted into an arm vein for blood sampling and will remain in the arm for the duration of the resting and exercise trials. Dr. Brent Ruby, a trained phlebotomist, will insert the indwelling catheter. Samples of 5 mL (about 1 tsp.) will be taken at rest and every 15 minutes post-exercise until blood glucose returns to normal. The total amount of blood sampled for each trial will not exceed 70 mL (about 14 tsp.). The total amount taken over the course of the entire study will not exceed 210 mL (about 42 tsp.). The blood samples will not be used for any other purpose in this study and any excess will be discarded.

Risks/Discomforts:

- All physical activity is accompanied by minimal risk.
- Mild discomfort such as muscle soreness, cramping, or shortness of breath may occur during or after each exercise trial.
- You will be asked periodically during testing to report any unusual symptoms. Unusual symptoms may include: undue shortness of breath, chest/jaw/arm/shoulder/upper back pain, dizziness, or any discomfort that differs from your normal exercise experience.
- Blood sampling can sometimes be associated with risks of bruising (10%), infection (less than 1%), and clotting problems (less than 1%). These risks will be minimized by use of sterile procedures and trained technicians. Signs and symptoms of infection at the catheter site include: redness, streaking, swelling, pain, and fever. If any of

these occur, you should contact Dr. Brent Ruby (243-2117 - office, 542-2513 - home). You will then be directed to seek medical care and the necessary treatment.

Benefits:

- There is no immediate benefit for taking part in this research study. However, your participation may aid health professionals (physicians, nurses, exercise physiologists, cardiac rehab specialists, personal trainers, etc.) in designing and prescribing a more beneficial exercise training intensity for patients with type II diabetes.

Confidentiality:

- Your identity will be kept confidential.
- When the results of this study are written in a scientific journal or presented at a scientific meeting, your name will not be used.

Compensation of Injury:

- Although we believe that the risk of taking part in this study is minimal, the following liability statement is required in all University of Montana consent forms.
In the event that you are injured as a result of this research you should individually seek appropriate medical treatment. If the injury is caused by the negligence of the University or any of its employees, you may be entitled to reimbursement or compensation pursuant to the Comprehensive State Insurance Plan established by the event of a claim of such injury, further information may be obtained from the University's Claims representative or University Legal Counsel.
(Reviewed by the University Legal Counsel, July 6, 1993)

Voluntary Participation/Withdrawal:

- Your decision to participate in this research study is entirely voluntary.
- You may withdraw from participation at any time.
- You may be asked to discontinue participation in the study if you fail to follow the instructions of the study director or if the study director believes it is in the best interest of your health and welfare.

Questions:

- If you have any questions concerning this research study contact: Brent Ruby, Ph. D. (Office: 243-2117, Home: 542-2513) or Andrew Miller (Office: 243-5528, Home: 549-8126) at the University of Montana Department of Health and Human Performance.
- If you have any questions regarding your rights as a research subject, you may contact the Institutional Review Board through the Research Office at The University of Montana at 406-243-6670.

Subject Statement of Consent:

I have read and understand the above description of this research study. I have been informed of the risk and benefits involved, and all my questions have been answered to my satisfaction. Furthermore, I have been assured that any future questions may be directed to a member of the research team. I voluntarily agree to participate in this study. I understand I will receive a copy of this consent form.

Name of subject (print)

Subject signature

Date

Signature of witness

Date

Medical and Resistance Training History Form

- 1. Do you have any previous history, or do you currently have type I or type II diabetes?**
- 2. Have you currently been engaged in a resistance training program for at least one month? If so, please describe the program you are currently participating in (days/week, length of session, etc.).**
- 3. Approximately how many years of resistance training experience do you have?**
- 4. Do you have any bleeding disorders that you are aware of?**

Physical Activity Readiness
Questionnaire - PAR-Q
(revised 1994)

PAR - Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	
☐	☐	1. Has your doctor ever said that you have a heart condition <u>and</u> that you should only do physical activity recommended by a doctor?
☐	☐	2. Do you feel pain in your chest when you do physical activity?
☐	☐	3. In the past month, have you had chest pain when you were not doing physical activity?
☐	☐	4. Do you lose your balance because of dizziness or do you ever lose consciousness?
☐	☐	5. Do you have a bone or joint problem that could be made worse by a change in your physical activity?
☐	☐	6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
☐	☐	7. Do you know of <u>any other reason</u> why you should not do physical activity?

If
you
answered

YES to one or more questions

Talk with your doctor by phone or in person **BEFORE** you start becoming much more physically active or **BEFORE** you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active — begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal — this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively.

DELAY BECOMING MUCH MORE ACTIVE:

- if you are not feeling well because of a temporary illness such as a cold or a fever — wait until you feel better; or
- if you are or may be pregnant — talk to your doctor before you start becoming more active.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

You are encouraged to copy the PAR-Q but only if you use the entire form

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction.

NAME _____

SIGNATURE _____

DATE _____

SIGNATURE OF PARENT
or GUARDIAN (for participants under the age of majority) _____

WITNESS _____

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Société canadienne de physiologie de l'exercice

Supported by:

Figure 3.2 PAR-Q pre-exercise screening test.

Appendix II:

Data Sheets

Exercise Trial Sheet

Date															
Subject #		Lever Arm Length										Total Trial Time			
HLV Trial (10 sets of 5)															
Right Leg												Left Leg			
	Mean Peak Force(N)		Total Work(J)		Running Total	Fatigue Index			Mean Peak Force(N)		Total Work(J)		Running Total	Fatigue Index	
	Quads	Hms	Quads	Hms		Quads	Hms		Quads	Hms	Quads	Hms		Quads	Hms
Set 1															
Set 2															
Set 3															
Set 4															
Set 5															
Set 6															
Set 7															
Set 8															
Set 9															
Set 10															
Set 11(opt)															
		Totals								Totals					
Date															
UHV Trial (5 sets of 15)															
Right Leg												Left Leg			
	Mean Peak Force(N)		Total Work(J)		Running Total	Fatigue Index			Mean Peak Force(N)		Total Work(J)		Running Total	Fatigue Index	
	Quads	Hms	Quads	Hms		Quads	Hms		Quads	Hms	Quads	Hms		Quads	Hms
Set 1															
Set 2															
Set 3															
Set 4															
Set 5															
Set 6(opt)															
		Totals								Totals					
Date															
1RM Test (N)		Right Leg		Left Leg											
	Quads	Hms	Quads	Hms											
60 deg/sec															
180 deg/sec															

Glucose and Insulin Sheet

Glucose											
Time	201-CT	202-CT	203-CT	204-CT	205-CT	206-CT	207-CT	208-CT	209-CT	210-CT	Means
0											
15											
30											
45											
Time	201-LIHV	202-LIHV	203-LIHV	204-LIHV	205-LIHV	206-LIHV	207-LIHV	208-LIHV	209-LIHV	210-LIHV	
0											
15											
30											
45											
Time	201-HILV	202-HILV	203-HILV	204-HILV	205-HILV	206-HILV	207-HILV	208-HILV	209-HILV	210-HILV	
0											
15											
30											
45											

Insulin											
Time	201-CT	202-CT	203-CT	204-CT	205-CT	206-CT	207-CT	208-CT	209-CT	210-CT	Means
0											
15											
30											
45											
Time	201-LIHV	202-LIHV	203-LIHV	204-LIHV	205-LIHV	206-LIHV	207-LIHV	208-LIHV	209-LIHV	210-LIHV	
0											
15											
30											
45											
Time	201-HILV	202-HILV	203-HILV	204-HILV	205-HILV	206-HILV	207-HILV	208-HILV	209-HILV	210-HILV	
0											
15											
30											
45											

Appendix III:

Statistical Results

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Subject	9	19.305	2.145				
trial	1	13.795	13.795	14.179	.0044	.0044	.0044
trial * Subject	9	8.756	.973				
TIME	3	51.476	17.159	24.965	.0001	.0001	.0001
TIME * Subject	27	18.557	.687				
trial * TIME	3	4.468	1.489	3.612	.0259	.0506	.0369
trial * TIME * Subject	27	11.132	.412				

Dependent: GLUCOSE

Comparison 1

Effect: trial * TIME
Dependent: GLUCOSE

	Cell Weight
LIHV, PRE	1.000
LIHV, 15	-1.000

df	1
Sum of Squares	14.146
Mean Square	14.146
F-Value	34.308
P-Value	.0001
G-G	.0001
H-F	.0001

Comparison 2

Effect: trial * TIME
Dependent: GLUCOSE

	Cell Weight
LIHV, PRE	1.000
LIHV, 30	-1.000

df	1
Sum of Squares	31.150
Mean Square	31.150
F-Value	75.550
P-Value	.0001
G-G	.0001
H-F	.0001

Comparison 3

Effect: trial * TIME
Dependent: GLUCOSE

	Cell Weight
LIHV, PRE	1.000
LIHV, 45	-1.000

df	1
Sum of Squares	8.295
Mean Square	8.295
F-Value	20.118
P-Value	.0001
G-G	.0011
H-F	.0004

Comparison 4
Effect: trial * TIME
Dependent: GLUCOSE

	Cell Weight
HILV, PRE	1.000
HILV, 15	-1.000

df 1
Sum of Squares 16.671
Mean Square 16.671
F-Value 40.434
P-Value .0001
G-G .0001
H-F .0001

Comparison 5
Effect: trial * TIME
Dependent: GLUCOSE

	Cell Weight
HILV, PRE	1.000
HILV, 30	-1.000

df 1
Sum of Squares 12.832
Mean Square 12.832
F-Value 31.122
P-Value .0001
G-G .0002
H-F .0001

Comparison 6
Effect: trial * TIME
Dependent: GLUCOSE

	Cell Weight
HILV, PRE	1.000
HILV, 45	-1.000

df 1
Sum of Squares 1.004
Mean Square 1.004
F-Value 2.434
P-Value .1304
G-G .1394
H-F .1360

Comparison 7
Effect: trial * TIME
Dependent: GLUCOSE

	Cell Weight
LIHV, PRE	1.000
HILV, PRE	-1.000

df 1
Sum of Squares .937
Mean Square .937
F-Value 2.274
P-Value .1432
G-G .1498
H-F .1477

Comparison 8
Effect: trial * TIME
Dependent: GLUCOSE

	Cell Weight
LIHV, 15	1.000
HILV, 15	-1.000

df 1
Sum of Squares .418
Mean Square .418
F-Value 1.013
P-Value .3232
G-G .2869
H-F .3063

Comparison 9
Effect: trial * TIME
Dependent: GLUCOSE

	Cell Weight
LIHV, 30	1.000
HILV, 30	-1.000

df 1
Sum of Squares 8.805
Mean Square 8.805
F-Value 21.354
P-Value .0001
G-G .0009
H-F .0003

Comparison 10
Effect: trial * TIME
Dependent: GLUCOSE

	Cell Weight
LIHV, 45	1.000
HILV, 45	-1.000

df 1
Sum of Squares 8.103
Mean Square 8.103
F-Value 19.652
P-Value .0001
G-G .0012
H-F .0004

Type III Sums of Squares

Source	df	Sum of Squares	Mean Square	F-Value	P-Value	G-G	H-F
Subject	9	88549.999	9838.889				
trial	1	7318.373	7318.373	2.483	.1495	.1495	.1495
trial * Subject	9	26527.591	2947.510				
TIME	3	51291.212	17097.071	13.804	.0001	.0012	.0007
TIME * Subject	27	33441.321	1238.567				
trial * TIME	3	9907.411	3302.470	4.841	.0080	.0344	.0276
trial * TIME * Subject	27	18419.030	682.186				

Dependent: INSULIN

	Count	Mean	Std. Dev.	Std. Error
LIHV, PRE	10	20.019	11.124	3.518
LIHV, 15	10	57.507	38.190	12.077
LIHV, 30	10	103.395	84.581	26.747
LIHV, 45	10	102.023	61.970	19.597
HILV, PRE	10	21.759	15.323	4.846
HILV, 15	10	60.685	38.412	12.147
HILV, 30	10	70.303	48.559	15.356
HILV, 45	10	53.681	43.634	13.798

Comparison 1
Effect: trial * TIME
Dependent: INSULIN

	Cell Weight
LIHV, PRE	1.000
LIHV, 15	-1.000

df	1
Sum of Squares	7026.751
Mean Square	7026.751
F-Value	10.300
P-Value	.0034
G-G	.0162
H-F	.0129

Comparison 2
Effect: trial * TIME
Dependent: INSULIN

	Cell Weight
LIHV, PRE	1.000
LIHV, 30	-1.000

df	1
Sum of Squares	34757.787
Mean Square	34757.787
F-Value	50.951
P-Value	.0001
G-G	.0001
H-F	.0001

Comparison 3
Effect: trial * TIME
Dependent: INSULIN

	Cell Weight
LIHV, PRE	1.000
LIHV, 45	-1.000

df	1
Sum of Squares	33623.280
Mean Square	33623.280
F-Value	49.288
P-Value	.0001
G-G	.0001
H-F	.0001

Comparison 4
Effect: trial * TIME
Dependent: INSULIN

	Cell Weight
HILV, PRE	1.000
HILV, 15	-1.000

df	1
Sum of Squares	7576.167
Mean Square	7576.167
F-Value	11.106
P-Value	.0025
G-G	.0136
H-F	.0106

Comparison 5
Effect: trial * TIME
Dependent: INSULIN

	Cell Weight
HILV, PRE	1.000
HILV, 30	-1.000

df	1
Sum of Squares	11782.600
Mean Square	11782.600
F-Value	17.272
P-Value	.0003
G-G	.0042
H-F	.0029

Comparison 6
Effect: trial * TIME
Dependent: INSULIN

	Cell Weight
HILV, PRE	1.000
HILV, 45	-1.000

df	1
Sum of Squares	5095.070
Mean Square	5095.070
F-Value	7.469
P-Value	.0109
G-G	.0312
H-F	.0268

Comparison 7
Effect: trial * TIME
Dependent: INSULIN

	Cell Weight
LIHV, PRE	1.000
HILV, PRE	-1.000

df	1
Sum of Squares	15.138
Mean Square	15.138
F-Value	.022
P-Value	.8827
G-G	.7001
H-F	.7408

Comparison 8
Effect: trial * TIME
Dependent: INSULIN

	Cell Weight
LIHV, 15	1.000
HILV, 15	-1.000

df	1
Sum of Squares	50.498
Mean Square	50.498
F-Value	.074
P-Value	.7876
G-G	.5971
H-F	.6361

Comparison 9
Effect: trial * TIME
Dependent: INSULIN

	Cell Weight
LIHV, 30	1.000
HILV, 30	-1.000

df	1
Sum of Squares	5475.402
Mean Square	5475.402
F-Value	8.026
P-Value	.0086
G-G	.0272
H-F	.0230

Comparison 10
Effect: trial * TIME
Dependent: INSULIN

	Cell Weight
LIHV, 45	1.000
HILV, 45	-1.000

df	1
Sum of Squares	11684.74
Mean Square	11684.74
F-Value	17.128
P-Value	.0003
G-G	.0044
H-F	.0029

Appendix IV:

Manuscript prepared for the Journal of Applied Physiology

**Effects of high intensity / low volume and low intensity / high volume isokinetic
resistance exercise on post-exercise glucose tolerance**

Running head: glucose tolerance after high and low intensity resistance exercise

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Abstract

The purpose of this study was to determine the effects of high intensity/low volume (HILV) and low intensity/high volume (LIHV) isokinetic resistance exercise on post-exercise glucose tolerance. Subjects ($n = 10$) performed two separate isokinetic resistance exercise trials (HILV and LIHV) of reciprocal concentric knee flexion and knee extension in a fasted state. Each bout was followed by a 45-min oral glucose ($1.8\text{g}\cdot\text{kg FFM}^{-1}$) tolerance test (OGTT). Blood samples were obtained every 15 min to determine glucose and insulin concentrations. There was no difference in total work (J) between the two trials ($p = .229$). Blood glucose was significantly higher at all time points compared to time 0 following the LIHV trial ($p < 0.05$). Following the HILV trial, blood glucose was significantly elevated at 15 and 30 min ($p < 0.05$) but returned to resting values by 45 min. Insulin concentration was significantly elevated following both trials at all time points ($p < 0.05$). Blood glucose and insulin were significantly higher following the LIHV at 30 and 45 min compared to the HILV trial ($p < 0.05$). These results demonstrate that although the total work output was similar across trials, high intensity muscle contraction is associated with an enhanced normalization of glucose homeostasis following a large post-exercise oral glucose feed.

Key Words

Resistance exercise, Glucose tolerance, Insulin, GLUT-4

Introduction

The physiological benefits of resistance training include improvements in muscular strength (5, 16, 20, 25, 31, 32, 42), bone mineral density (42), blood lipid profiles (24, 25), and fat free mass (FFM) (32). Resistance training has also been shown to maintain FFM during caloric restriction (14, 36), improve endurance performance (31), and glycemic control (16,17, 24, 32).

Several studies have demonstrated increased glucose uptake and subsequent muscle glycogen resynthesis following resistance exercise (33, 37, 38). Roy et al. (38) stated that the increased muscle glycogen resynthesis was most likely a result of increased GLUT-4 translocation. Therefore, it is possible that resistance exercise may improve glycemic control in patients with insulin resistance. The pathology behind type II diabetes involves both peripheral insulin resistance and a decreased insulin secretion thus resulting in hyperglycemia. Eriksson et al. (17) and Honkola et al. (24) demonstrated improvements in long-term glycemic control (glycosylated hemoglobin) following a circuit resistance training program in type II diabetics. Similarly, Durak et al. (16) showed improvements in glycemic control in type I diabetics following a resistance training program. Other authors have noted a reduction in the plasma insulin response to an oral glucose load following the completion of a resistance training program (25, 32). These data suggest that the normal adaptations to resistance exercise may include an improved glycemic control in clinical populations with insulin resistance. However, the effects of contractile intensity during resistance exercise on glycemic control have not been addressed.

Three review papers by Barnard and Youngren (6), Borghouts and Keizer (10), and Hayashi et al. (23) suggest that the process of muscle contraction results in GLUT-4 translocation to the plasma membrane and transverse tubules independent of insulin release or binding. It is evident that contraction-induced glucose transport is proportionate to the magnitude of GLUT-4 translocation (39). Other studies using rats have demonstrated similar findings (13, 22, 34). In contrast, a single bout of eccentric exercise is associated with a decrease in GLUT-4 content (2, 3, 4). It has also been suggested that two distinct intracellular locations of glucose transporters exist, one that responds to exercise and one that responds to insulin (13, 18, 28, 43). Therefore, the stimulation of GLUT-4 translocation from both pools may induce a maximal effect on glucose uptake and the subsequent rates of glycogen resynthesis.

Several studies have investigated post-exercise glucose uptake immediately following continuous endurance exercise (7, 8, 9, 26, 27, 29, 30, 35, 45). Furthermore, studies by Bonen et al. (11), Braun et al. (12), and Young et al. (44) compared the effects of exercise intensity on glucose uptake. All three of the studies found no difference in blood glucose and insulin responses between high and low intensity cardiovascular exercise. In contrast, few studies have examined glucose uptake following resistance exercise (33, 37, 38) whereas to our knowledge, none have investigated the effect of resistance exercise intensity on post-exercise glycemic control. Therefore, the purpose of this study was to determine the effects of high intensity/low volume (HILV) and low intensity/high volume (LIHV) isokinetic resistance exercise on post-exercise glucose tolerance. We hypothesized that, when total work was held constant, higher intensity contractile force

would result in a more rapid normalization of blood glucose and insulin following a post-exercise oral feeding of carbohydrate.

Methods

Subjects

A total of 10 college-aged males served as subjects for the study. Each subject had at least one year of recreational resistance training experience and had currently been involved in a resistance-training program for a minimum of one month. Questionnaires regarding subjects' previous resistance training experience, medical history, and physical activity levels were assessed before testing commenced. Prior to participation, each subject completed a University IRB-approved informed consent form.

Descriptive Data

Descriptive data including height, weight, age, body composition, and previous resistance training experience were collected. Height (cm) was determined using a conventional stadiometer. Weight was measured with a calibrated digital scale (Toledo Model 8139, Worthington, OH). Body composition, fat mass (FM), and fat free mass (FFM) was assessed by hydrodensitometry at residual lung volume. Subjects performed repeated underwater weight trials until three values within 100 grams were obtained. Residual lung volume was calculated using a Helium dilution technique outside of the hydrostatic weighing tank in a seated position. Subjects performed at least three trials where the two closest values within 250 mL were averaged. Body density was converted to percent body fat using the Siri equation (40).

Exercise Testing

All muscle testing was performed using the Kinetic Communicator (KIN-COM Model #125AP, Chattecx Corp. Chattanooga, TN), a computer controlled isokinetic dynamometer. Each subject participated in a session to determine Average Peak Force (Newtons (N)) during isokinetic movements at speeds of 60° and 180°/sec through a range of 80° (knee extension through flexion). Prior to testing, subjects were allowed to warm up on a cycle ergometer at a self-selected pace for approximately 5 min.

Goniometry was used to acquire the desired range of motion. Subjects performed the Average Peak Force test first at 60°/sec then at 180°/sec. On both extremities, subjects performed 2 sets of 5 and 1 set of 3 repetitions: one set at approximately 50% of maximal effort, one set at approximately 75% of maximal effort, and the final set at 100% of maximal effort. Subjects were allowed full recovery between sets. For the speeds of 60°/sec and 180°/sec, the Average Peak Force (N) of 3 repetitions for leg extension and flexion of the final set was used as the Average Peak Force for both leg extension and flexion. For data analysis the absolute Average Peak Force for each subject was converted into a relative value by dividing the Average Peak Force by their FFM.

Each subject participated in two isokinetic resistance exercise trials. The high intensity/low volume exercise trial (HILV) consisted of subjects performing 10 sets of 5 repetitions of reciprocal concentric knee extension / knee flexion exercise. Prior to the trial, subjects were allowed to warm up on a cycle ergometer at a self-selected pace for approximately 5 min. One repetition was considered a full concentric extension and concentric flexion of the knee joint within a range of 80°. Speed of the motion was set at

60°/sec with 2-min rest intervals between sets. Subjects completed 5 sets on each extremity in an alternating manner until 10 sets was reached. Approximately 4-5 min was permitted when alternating extremities. Selection of the extremity to perform the exercise first was done randomly. Total work in joules (J) was provided by the KIN-COM after completion of each set and the sum was calculated manually to quantify total work (J) for the entire session. In addition, the Mean Peak Force from each set was averaged to determine the Mean Peak Force for the entire session for knee extension and flexion on both extremities. For data analysis the absolute Mean Peak Force for each subject was converted into a relative value by dividing the Mean Peak Force by their FFM.

The low intensity/high volume exercise trial (LIHV) included identical components as the HILV trial with the following exceptions. Subjects performed 4 sets of 15 concentric knee extension / knee flexion exercise. Speed of the motion was set at 180°/sec with 2-min rest intervals between sets and 4 to 5 min when alternating extremities.

Order of the trials

The HILV and LIHV trials were conducted at least 4 days apart in random order. Subjects were asked to refrain from strenuous exercise 24 hours prior to the two trials. The total work from either the HILV or LIHV trial, whichever was performed first, was used as a stopping point for the following trial to be completed. This was done to normalize the total work between trials.

Oral Glucose Tolerance Test's (OGTT)

Prior to each trial, subjects reported to the lab following a 12-hour fast. An indwelling venous catheter was then inserted into an antecubital vein and kept patent using a continuous saline drip. The time = 0 sample was collected upon insertion of the indwelling line. Each subject was then transported to the Outpatient Physical Therapy Clinic at St. Patrick's Hospital and Health Sciences Center to complete one of the two exercise trials in random order. Immediately post-exercise, a blood sample was taken followed by consumption of the dextrose drink. Once the drink was finished, the time for the OGTT was started. Subjects completed the OGTT ($1.8\text{g}\cdot\text{kg FFM}^{-1}$, 20% dextrose drink) immediately following both the HILV and LIHV exercise trials, lasting 45 minutes.

Blood Sampling

Blood samples (~5 mL) were collected every 15 min in untreated test tubes and allowed to clot for 15 min at room temperature. Samples were spun at approximately 4000 rpm at 4°C for 15 min after which the plasma was separated from the whole blood. Samples were stored at -30°C until subsequent analysis.

Metabolite assays

Blood glucose concentration was determined in duplicate using a spectrophotometric technique using a commercially available kit (Procedure UV-47, Sigma Diagnostics, St. Louis, MO). Blood insulin was determined in duplicate using a solid phase two-site enzyme immunoassay (DRG Insulin ELISA, DRG International, Inc., USA).

Statistical analyses

All descriptive data for subjects is presented in terms of mean $\pm$ SD. A Bland Altman Plot and paired samples t-test was used to compare total work (J) between the HILV and LIHV trials. A paired samples t-test of means was used to compare Average Peak Force (N/FFM (kg)) and Mean Peak Force (N/FFM (kg)). A repeated measures 2 x 4 design ANOVA was used to examine differences across time (0, 15, 30, and 45 min post-exercise) and trial (HILV and LIHV). All data analyses were conducted using Microsoft Excel and the SuperAnova statistical package (Abacus Inc, Berkeley, CA). The level of significance was set at an alpha level of 0.05.

Results

Descriptive statistics including age (years), height (cm), weight (kg), percent body fat, fat free mass (kg), fat mass (kg) and g of carbohydrate (CHO) consumed for the subjects used in the study are provided in Table 1. Average Peak Force (N/FFM (kg)) and Mean Peak Force (N/FFM (kg)) are provided in Table 2. For both Average Peak Force and Mean Peak Force no significant difference was found between the left and right sides, therefore the data was collapsed and is reported by muscle group only. For the Average

Peak Force test, significant differences were found between the two intensities (60 deg/sec and 180 deg/sec) for the knee extensors and knee flexors ($p < 0.05$). Significant differences were found for Mean Peak Force between the LIHV and HILV trials for knee extension, but not for flexion of either extremity ($p < 0.05$).

The total work (J) completed for the LIHV and HILV trials is provided in Table 3. Total work for the LIHV and HILV trials was 25784.3 ± 4320.6 J and 25882.1 ± 4439.4 J respectively. The difference in total work between the two trials was not significant ($p = 0.229$). Figure 3 is a Bland Altman Plot showing the difference in total work versus the average total work for both trials. 90% of subjects had less than 1% difference in total work between the LIHV and HILV trials. The remaining 10% had less than 3% difference between trials.

Figures 1 and 2 demonstrate the blood glucose and insulin responses respectively for the time points 0, 15, 30, and 45 min post-exercise. A significant time x trial interaction was found for both blood glucose ($p = .03$) and insulin ($p = .01$). Blood glucose was significantly higher from time = 0 at all time points during the LIHV trial ($p < 0.05$). During the HILV trial, blood glucose was significantly elevated from time = 0 at 15 and 30 min ($p < 0.05$) but returned to resting values by 45 min. Insulin concentration was significantly elevated from time = 0 min at all time points for both trials ($p < 0.05$). Blood glucose and insulin were significantly higher during the LIHV at time = 30 and 45 min compared to the HILV trial ($p < 0.05$).

Discussion

The purpose of this investigation was to determine the effects of high and low intensity resistance exercise on post-exercise glucose tolerance when the total muscle work was kept constant. Results of the study demonstrate an enhanced normalization of blood glucose and insulin following the HILV trial. This is of significance considering the similarity in total work between trials (see Figure 3). The HILV trial was designed to mirror the common prescription for the development of muscular strength including less than 6 repetitions per set performed at a slower speed.

The only previous research examining the effects of exercise intensity on glucose tolerance and insulin responses has used an endurance exercise protocol (11, 12, 44). One study found no differences between intensities in glucose and insulin areas under the curve both immediately after exercise and 24-hours post-exercise (11). A possible limitation to this study is that the authors had subjects exercise at intensities of 60 and 83% of age-adjusted HR_{max} for the low and high intensity exercise trials. The variance associated with prescribing cardiovascular exercise from heart rates is considerable (1 S.D. = 10-12 b/min) (1). In addition, the high intensity session was performed one week prior to the low intensity session thus possibly causing a residual effect of the previous exercise session.

A study by Young et al. (44) used trained and non-trained subjects and found no differences in plasma glucose and insulin responses to an oral glucose load 14 hours post-exercise at intensities of 40 and 80% of VO_{2max}. However, after a single bout of exercise,

the non-trained subjects had comparable insulin responses to the trained subjects. Braun et al. (12) had women with non-insulin-dependent diabetes mellitus (NIDDM) exercise at 50 and 75% of $\text{VO}_{2\text{max}}$ and adjusted the duration of exercise so that energy expenditure was equal in both exercise conditions. No differences were found when comparing plasma glucose and insulin responses to a test meal (50% carbohydrate, 20% protein, and 30% fat) 90 min post-exercise.

It has been demonstrated that exercise/muscle contraction mediated glucose transport is proportionate to the magnitude of GLUT-4 translocation to the plasma membrane. Some studies have shown that insulin plus contraction increases glucose uptake above that of insulin or contraction alone (13, 22, 34) whereas others have not (15, 21). However, Constable et al. (15) used a nonphysiological high insulin concentration (20,000 $\mu\text{U/ml}$ for 60 min) to elucidate the actions of insulin. The mechanism by which muscle contraction may activate glucose transport appears to involve the initiation of a contraction signal. Possible reasons for this initiation could include: release of calcium, activation of AMP-activated protein kinase, and nitric oxide activity (23, 39). An explanation for the improved glucose tolerance and insulin response following the HILV trial could be the magnitude of muscle contraction involved. During the HILV exercise trial the subjects were working their knee extensors at an intensity that was significantly greater than during the LIHV exercise trial. Interestingly, there was no significant difference in the work intensity of the knee flexors between the HILV and LIHV exercise trials (see Table 2). Thus, the magnitude of contraction of the knee extensors that occurred may result in a greater GLUT-4 translocation and subsequent glucose transport.

These data indicate that the intensity of contraction is more influential on glucose normalization in comparison to the total muscle work. In addition, a possible link may exist between fast glycolytic fibers and subsequent GLUT-4 translocation. Thus, the improvement in glucose homeostasis shown may be a result of higher intensity contractile force and/or increased fast glycolytic fiber recruitment.

A possible limitation to the study may have been the duration of exercise and rest for each trial. Total exercise and rest time for the HILV trial was 3.8 ± 0.8 and 46.0 ± 7.9 min respectively where total exercise and rest time for the LIHV trial was 2.1 ± 0.4 and 22.8 ± 4.4 min respectively. The difference between the two trials was due to a higher number of sets and rest periods associated with the HILV trial.

The study may have implications towards individuals with insulin resistance or deficiency such as is the case with Type II diabetes. Resistance exercise has already been shown to improve the metabolic profile through either improved glucose tolerance and/or insulin response in people with Type I (16) or Type II diabetes (17, 19, 24, 41). These results demonstrate both improved glucose tolerance and insulin response following a resistance exercise bout similar to the standard for strength development. Therefore, the benefits of higher intensity and lower volume exercise should receive further attention. The effects of fast glycolytic fiber recruitment strategies should also be investigated in terms of the potential to maximize GLUT-4 translocation. In addition, the link between total fiber recruitment and/or fiber type recruitment and GLUT-4 translocation should be addressed. Interestingly, these results conflict with the guidelines set by the American

College of Sports Medicine, which recommends performing resistance exercise in the range of 8 to 12 repetitions (1). Furthermore, these data suggest that decisions regarding the amount of post-exercise carbohydrate supplement are dependent on the nature of the resistance exercise bout (high vs. low intensity) during strength training.

In conclusion, to our knowledge this is the first study to examine blood glucose and insulin levels following two different resistance exercise trials when the total work between the two trials was kept constant. The results demonstrate a more rapid blood glucose normalization in addition to a blunted insulin response following a bout of high intensity / low volume isokinetic resistance exercise. This is most likely a result of enhanced stimulation of contraction sensitive GLUT-4 transporters to the plasma membrane and may indicate fiber dependent and/or ramp GLUT-4 recruitment process during resistance exercise. Further research is needed examining metabolic responses following resistance exercise when variables such as the duration of exercise, mode of resistance exercise, active muscle mass, and type of subjects used are manipulated.

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Table 1. Physical characteristics of subjects.

Age (years)	21.5 ± 2.0
Height (cm)	179.9 ± 7.7
Weight (kg)	13.6 ± 6.3
Body fat %	15.2 ± 5.0
FFM (kg)	72.5 ± 8.1
FM (kg)	13.6 ± 6.3
g CHO consumed	130.5 ± 14.5

Data expressed as means ± S.D. FFM, fat free mass; FM, fat mass.

Table 2. Comparisons of Average Peak Force (N/FFM (kg)) and Mean Peak Force (N/FFM (kg)) between LIHV (180 deg/sec) and HILV (60 deg/sec) trials on the KIN-COM dynamometer.

Average Peak Force	LIHV	HILV
Knee Extensors	7.70 ± 1.04	10.04 ± 1.21*
Knee Flexors	4.72 ± 0.78	5.05 ± 0.60*
Mean Peak Force	LIHV	HILV
Knee Extensors	6.00 ± 0.64	9.24 ± 1.41*
Knee Flexors	5.27 ± 0.76	5.52 ± 0.89

Data expressed as means ± S.D. * Significantly different between intensities $p < 0.05$.

Table 3. Total Work (J) between LIHV (180 deg/sec) and HILV 60 deg/sec) trials.

	LIHV	HILV
Right Leg	12868.7 ± 2427.4	12911.1 ± 2443.9
Left Leg	12915.7 ± 1954.8	12971.0 ± 2061.8
Total	25784.3 ± 4320.6	25882.1 ± 4439.4

Data expressed as means ± S.D. * Significantly different between intensities p < 0.05.

Figure Legend

- Figure 1.** Plasma glucose responses (mmol) during an OGTT following the HILV and LIHV exercise trails. Time = 0 was started immediately post-exercise after consumption of standard glucose drink. Data expressed as mean $\pm$ standard error. ^aSignificantly different from time = 0 for HILV trial, $p < 0.05$. ^bSignificantly different from time = 0 for LIHV trial, $p < 0.05$. ^cSignificantly different from LIHV trial, $p < 0.05$.
- Figure 2.** Plasma insulin responses (μ IU/ml) during an OGTT following the HILV and LIHV exercise trails. Time = 0 was started immediately post-exercise after consumption of standard glucose drink. Data expressed as mean $\pm$ standard error. ^aSignificantly different from time = 0 for HILV trial, $p < 0.05$. ^bSignificantly different from time = 0 for LIHV trial, $p < 0.05$. ^cSignificantly different from LIHV trial, $p < 0.05$.
- Figure 3.** A Bland Altman Plot showing the Difference in total work (J) vs. Average total work (J). $p = .229$.